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# THE INFLUENCE OF A ZERO-CALORIE DIET ON CARDIAC PERFORMANCE IN OVERWEIGHT WOMEN WITH NORMAL CIRCULATORY SYSTEMS

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Translation of: "Der Einfluss einer Null-Kaloriendiät auf die Herzleistung von kreislaufgesunden, übergewichtigen Frauen," in Wiener Klinische Wochenschrift, vol. 85, Oct. 1973, pp. 657-661.





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16. Abstract

Fasts of two weeks are employed to reduce overweight in 9 obese, but otherwise healthy women. Before treatment and at the end of the first and second week of total starvation, serum electrolytes ($K^+$, $Na^+$, $Ca^{++}$, $Cl^-$), total serum protein, and acid-base status were measured. The ejection fraction and index of myocardial contractility were calculated based on systolic time intervals. During starvation significant reduction in left ventricular ejection time was observed as well as prolongation of pre-ejection period and external isovolumic contraction time. After two weeks significant reduction in ejection fraction occurred and the index of myocardial contractility was significantly depressed. The serum $K^+$ concentration shifted to lowered values. It is suggested that total therapeutic starvation of relatively short duration may cause reduction in myocardial contractility of the left ventricle. So that total starvation does not seem a harmless approach and needs close supervision of cardiac action.

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# THE INFLUENCE OF A ZERO-CALORIE DIET ON CARDIAC PERFORMANCE IN OVERWEIGHT WOMEN WITH NORMAL CIRCULATORY SYSTEMS

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Overweight leads to a series of resulting illnesses and presents thereby a serious clinical problem. The most effective method of treatment of overweight consists of a complete restriction of calories, the so-called Zero-Calorie Diet. As early as 1915, Benedict [3] reported on the material change processes during a 30-day fasting period in a patient. Bloom [4] used the method in 1959 in the hospital. Since then, this method of treatment of overweight has been used frequently, and appears generally to be relatively harmless. Even long-lasting periods of fasting were carried out in many cases without serious complications [5, 20, 22]. Generally, the unremarkable behavior of pulse, blood pressure, and electrocardiogram are given as evidence for the harmlessness of a zero-calorie diet with respect to the cardiovascular system. At our hospital, several circulatorily healthy overweight patients on a zero-calorie diet exhibited dizziness, nausea, and conditions of collapse after a short period of fasting, although at this time no EKG changes were detectable and also the blood pressure and pulse had not changed essentially. /657*

It seemed important to us, therefore, to carry out studies with these patients using a broadened methodology, in order to have a better understanding of the cardiac performance. In a

*Numbers in the margin indicate pagination in the foreign text.

series of studies, the usefulness of the systolic cycle time and the indices calculated from it could be demonstrated for judgment of the myocardian function [1, 10, 17, 21, 23, 24[. By the use of these methods now, a better insight into the hemodynamic situation of the patients should be obtained. /658

Methodology

Nine women aged from 16 to 50 years were studied, who all were clearly overweight. With the exception of the overweight condition, no pathologically clinical findings could be produced. The average deviation from average weight amounted to 53 ± 13.6% (29 to 68%). The overweight is stated as a percentage deviation from the average weight of the population, on the basis of the publications of the Society of Actuaries [6]. The patients were taken into confinement and a zero-calorie diet was imposed. During the fasting cure, the patients were forbidden bodily activity such as walking or gymnastics.

The zero-calorie diet was carried out in the following way: Each day the patients took two liters of mineral water, a multivitamin preparation, and one teaspoonful of Elo-oral (1 teaspoonful contains 25 mVal Sodium, 10 mVal Potassium, 2 mVal Calcium, 2 mVal Magnesium, 15 mVal Citrate, 2 mVal Lactate, 15 mVal Chloride, 2 mVal Sulfate, 5 mVal Phosphate) three times.

Before the beginning of the fasting cure, as well as on the 8th and 15th days after the beginning of the treatment, the following tests were carried out: determination of the serum concentration of potassium, sodium, calcium and chloride ions as well as the total protein. Measurement of the actual ph and of the base excess, according to the method of Sigaard-Andersen and Engel. Blood pressure measurement according to Korotkoff, simultaneous recording of the electrocardiogram, phonocardiogram, and carotid sphygmogram, by means of the Minograf 81 of the Elema-Schonander Firm. The measurement of the systolic cycle time was carried out according to the instructions of Holldack and Wolf [11]. The contractility index was calculated as the quotient of diastolic blood pressure and pressure rise time, according to Landry and Goodyer [15]. The expelled fraction was determined according to the instructions of Garrard and coworkers [10].

## Results

The individual data as well as the average values and variations of the measured and calculated parameters are set forth in Tables 1 through 3.

### Weight Reduction:

All studied persons lost weight significantly. The average weight loss after two weeks amounted to 7.39 $\pm$ 2.69 kg ($t = 8.211$, $p < 0.001$), of which the patients lost on the average, about 5.0 $\pm$ 2.3 kg in the first week, and 2.39 $\pm$ 0.96 kg in the second week.

### Serum Electrolytes:

While average $Na^+$, $Ca^{++}$, and $Cl^-$ concentrations in the serum changed practically not at all in our patients during the 14-day fasting period (Table 3), on the average the $K^+$ concentration decreased after two weeks of the diet. However, this decrease could not be statistically verified ($t = 2.258$, $p > 0.05$).

the total protein did not change at all, on the average, during the fasting period.

Pulse Frequency:

The pulse frequency (Table 1) climbed in the course of the first week from 67 ± 6 beats/min. to 81 ± 24 beats/min., yet this difference is not significant. At the end of the second week of therapy, the pulse frequency amounted to 85 ± 21 beats/min., and this value was significantly elevated compared to the initial value ($t = 2.521$, $p < 0.05$).

Average Blood Pressure:

The arterial average pressure (Table 1) fell after one week of the fasting cure from 106 ± 12 mm Hg to 96 ± 15 mm Hg, and after two weeks to 94 ± 12 mm Hg. Both the decrease after one week ($t = 3.689$, $p < 0.001$) and also after the second week ($t = 5.903$, $p < 0.001$), were significant.

Diastolic Blood Pressure:

The diastolic blood pressure (Table 1) fell in the first week from 87 ± 11 mm Hg to 78 ± 11 mm Hg, which is a significant finding ($t = 2.694$, $p < 0.05$), and remained practically the same in the course of the second week of treatment at 77 ± 10 mm Hg.

Cycle Time:

Pressure rise time: The pressure rise time (Table 2) before the beginning of therapy amounted to 25 ± 8 msec., at the end of the first week 26 ± 9 msec., and rose significantly only in the course of the second week of treatment ($t = 3.061$, $p < 0.01$) to 37 ± 13 msec. All these values, however, lie within the normal range.

Time of strain:

The time of strain (Table 2) lengthened in the first week, not significantly, from 97 $\pm$ 13 msec. to 102 $\pm$ 18 msec. ($t = 0.753$, $p < 0.1$). In the course of the second week, a significant lengthening to 116 $\pm$ 21 msec. took place ($t$ [illegible] 2.697, $p < 0.05$). At the end of the first week, three patients exhibited a lengthened time of strain for the existing pulse frequency, above the normal range, and at the end of the second week this was the case for 7 of 9 patients. The average value for the whole group at the end of the second week of treatment was likewise moderately lengthened in comparison with the existing pulse frequency.

Expulsion Time:

The expulsion time (Table 2) fell significantly in the course of the first week from 310 $\pm$ 16 msec. to 274 $\pm$ 42 msec. ($t = 2.673$, $p < 0.05$). In the course of the second week, a further reduction to 252 $\pm$ 47 msec. was determined. This last value differs significantly from the initial value ($t = 3.207$, $p < 0.02$), however it did not vary significantly from the value after one week ($t = 1.643$, $p < 0.1$). In comparison with the existing pulse frequency, 4 of 9 patients exhibited too short an expulsion time at the end of the first week, the rest of the patients had a normal value for the existing frequency, also, the average value for the entire group lay within the normal range with regard to the existing average pulse frequency. After the second week of therapy, the value was significantly below the suitable normal range for the existing pulse frequency in the case of five patients, and the average value for the whole group lay below this normal limit [1]].

Table 1

| Name | Age | Height (cm) | Relative Body Wt., % deviation from avg. wt. | Pulse Frequency (Beats per Minute) | | | Avg. Arterial Pressure (mm Hg) | | | Diastolic Blood Pressure (mm Hg) | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | before diet | after 1wk | after 2wk | before diet | after 1wk | after 2wk. | before diet | after 1wk | after 2wk |
| A. E. | 17 | 160 | 68.0 | 69 | 55 | 55 | 97 | 88 | 91 | 80 | 75 | 70 |
| F. K. | 33 | 164 | 56,5 | 67 | 70 | 86 | 100 | 92 | 92 | 85 | 70 | 70 |
| F. E. | 16 | 156 | 61,3 | 73 | 116 | 108 | 104 | 81 | 83 | 95 | 60 | 70 |
| G. E. | 49 | 167 | 40,5 | 70 | 66 | 67 | 115 | 101 | 97 | 100 | 80 | 80 |
| L. G. | 50 | 150 | 55,7 | 64 | 87 | 63 | 126 | 112 | 107 | 100 | 95 | 90 |
| M. H. | 24 | 170 | 39,0 | 59 | 53 | 100 | 94 | 90 | 86 | 70 | 75 | 75 |
| O. M. | 26 | 168 | 29,0 | 60 | 99 | 100 | 104 | 83 | 92 | 80 | 70 | 70 |
| S. G. | 44 | 166 | 62,2 | 77 | 114 | 111 | 123 | 125 | 119 | 95 | 95 | 95 |
| W. R. | 33 | 148 | 65,0 | 64 | 69 | 71 | 93 | 89 | 81 | 80 | 80 | 70 |
| $\bar{x}$ | | | 53.0 | 67 | 81 | 85 | 106 | 96 | 94 | 87 | 78 | 77 |
| s | | | 13,6 | 6 | 24 | 21 | 12 | 15 | 12 | 11 | 11 | 10 |

Table 2

| Name | % Expelled from left ventricle | | | Contractility Index (mm Hg/sec.) | | | Pressure Rise Time (msec.) | | | Time of Strain (msec.) | | | Expulsion Time (msec.) | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | before diet | after 1wk. | after 2wk. | before diet | after 1wk. | after 2wk. | before diet | after 1wk. | after 2wk. | before diet | after 1wk. | after 2wk. | before diet | after 1wk. | after 2wk. |
| A. E. | 75 | 69 | 72 | 5000 | 4420 | 3690 | 16 | 17 | 19 | 81 | 106 | 98 | 286 | 310 | 322 |
| F. K. | 78 | 71 | 63 | 3150 | 2910 | 1630 | 27 | 24 | 43 | 84 | 96 | 108 | 312 | 292 | 279 |
| F. E. | 65 | 75 | 56 | 3970 | 4620 | 2420 | 24 | 13 | 29 | 109 | 65 | 90 | 291 | 222 | 199 |
| G. E. | 74 | 70 | 64 | 4545 | 2210 | 1483 | 22 | 38 | 54 | 94 | 106 | 126 | 310 | 309 | 314 |
| L. G. | 74 | 63 | 43 | 2770 | 4520 | 1610 | 36 | 21 | 56 | 97 | 112 | 152 | 320 | 286 | 276 |
| M. H. | 75 | 72 | 31 | 3330 | 2581 | 1702 | 21 | 29 | 44 | 101 | 106 | 135 | 339 | 332 | 210 |
| O. M. | 72 | 53 | 51 | 2420 | 2695 | 2590 | 33 | 26 | 27 | 102 | 109 | 108 | 315 | 232 | 222 |
| S. G. | 75 | 62 | 51 | 5820 | 4120 | 3654 | 16 | 23 | 26 | 85 | 86 | 97 | 314 | 216 | 208 |
| W. R. | 63 | 50 | 46 | 2350 | 2000 | 1790 | 34 | 40 | 39 | 118 | 129 | 126 | 300 | 264 | 239 |
| $\bar{x}$ | 72 | 65 | 53 | 3706 | 3331 | 2285 | 25 | 26 | 37 | 97 | 102 | 116 | 310 | 274 | 252 |
| s | 5 | 9 | 12 | 1211 | 1077 | 630 | 8 | 9 | 13 | 13 | 13 | 21 | 16 | 42 | 47 |

Table 3

| Name | $K^+$ Concentration (milliequivalents per liter serum) | | | $Na^+$ Concentration | | | $Ca^{++}$ Concentration | | | $Cl^-$ Concentration | | | Total Protein Concentration (g-%) | | | Actual pH | | | Base Excess (meq/l blood) | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | before diet | after 1wk. | after 2wk. | before diet | after 1wk. | after 2wk. | before diet | after 1wk. | after 2wk. | before diet | after 1wk. | after 2wk. | before diet | after 1wk. | after 2wk. | before diet | after 1wk. | after 2wk. | before diet | after 1wk. | after 2wk. |
| A. E. | 4.3 | 4.3 | 3,9 | 136 | 136 | 140 | 5.20 | 4,90 | 4.84 | 108 | 102 | 110 | 7,2 | 7,5 | 7,0 | 7,39 | 7.31 | 7.27 | +0.3 | — 7.5 | —2.5 |
| F. K. | 4.1 | 4.3 | 4,5 | 144 | 138 | 137 | 4.72 | — | 4.96 | 104 | — | — | 7,6 | 7.7 | 7.0 | 7,38 | 7.31 | 7.30 | +1.5 | — 6.0 | —9,5 |
| F. E. | 4,0 | 4.0 | 3,7 | 138 | 140 | 134 | 4.80 | 4.70 | 4,96 | 106 | 99 | 99 | 6,8 | 7,4 | 7,7 | 7,37 | 7.31 | 7.34 | +0.5 | — 9,1 | —5,0 |
| G. E. | 4,1 | 4,0 | 4.6 | 136 | 140 | 139 | 4.60 | 5,12 | 4,90 | 113 | 100 | 100 | 7,2 | 7.4 | 7,3 | 7,44 | 7.32 | 7,40 | +1,0 | — 3.0 | —2.5 |
| L. G. | 4.4 | 3,8 | 3.8 | 134 | 138 | — | 4.60 | 4.80 | — | — | — | — | 6.3 | 6.8 | 6,3 | 7,40 | 7.36 | 7,34 | —2.0 | — | —9,9 |
| M. H. | 4.3 | 4.0 | 3.1 | 138 | 136 | — | 4,70 | 4,52 | — | 102 | 104 | — | 6.7 | 7,0 | — | 7.36 | 7.30 | 7.32 | +0.5 | —11.5 | — |
| O. M. | 4.0 | 3,7 | 3,2 | 138 | 136 | 132 | 4,88 | 5.10 | 4,96 | 109 | 98 | 91 | 6,3 | — | 7,7 | 7,44 | 7.42 | 7,44 | —1,0 | — 3.0 | —1,0 |
| S. G. | 4.1 | 4,4 | 3,6 | 138 | 136 | 136 | 4.20 | 4.80 | 4.60 | 102 | 99 | 96 | 7,3 | 7.5 | 7,3 | 7,47 | 7.36 | 7.43 | +1.0 | —10.0 | —1,2 |
| W. R. | 4,4 | 4.2 | 3.1 | 140 | 136 | 144 | 4.40 | 5,04 | 5,06 | 106 | 106 | 98 | 7.0 | 7,4 | 7,7 | 7,38 | 7,28 | 7.32 | —1,0 | —11,0 | —9,5 |
| $\bar{x}$ | 4.2 | 4.1 | 3,7 | 138 | 137 | 137 | 4.69 | 4.87 | 4.90 | 106 | 101 | 99 | 6.9 | 7.3 | 7.3 | 7,40 | 7,33 | 7.35 | +0,8 | — 7.6 | —5,1 |
| s | 0,16 | 0.24 | 0.56 | 2.8 | 1.7 | 4,0 | 0.29 | 0.21 | 0.15 | 4.3 | 3,0 | 5.7 | 0.47 | 0,29 | 0.48 | 0.05 | 0,05 | 0,06 | 1,13 | 3.37 | 3,66 |

Contractility Index:

The contractility index (Table 2) fell slightly after one week of fasting, from 3706 $\pm$ 1211 mm Hg/sec. to 3331 $\pm$ 1077 mm Hg/sec., however, this difference was not significant. After the second week, the average contractility index for the whole group lay statistically significantly lower at 2285 $\pm$ 630 mm Hg/sec., both compared to the initial value ($t = 4.670$, $p < 0.01$) and also compared to the value after one week of fasting ($t = 3.328$, $p < 0.02$).

Expelled Fraction: /660

The expelled fraction (Table 2) fell significantly on the average from 72 $\pm$ 5% to 65 $\pm$ 9% ($t = 2.665$, $p < 0.05$). However, both of these values lay within the normal range . In the course of the second week of treatment, the expelled fraction was reduced to 53 $\pm$ 12%, and thereby lay significantly lower compared with the initial value ($t = 6.034$, $p < 0.001$), and also compared with the value after one week ($t = 2.950$, $p < 0.02$).

Positive correlations were demonstrated between $K^+$ concentration and expelled fraction, as well as between $K^+$ concentration and expulsion time ($r = 0.668$, $p < 0.001$, or $r = 0.584$- $p < 0.01$). No correlation could be shown between the $K^+$ concentration and the excess circulatory parameters. If the decrease in $K^+$ concentration is set up in relation to the changes in the respective circulatory parameters, then no correlations could be found.

The EKG was in the normal range before the beginning of therapy as well as at the end of the first and second weeks all patients, and the heart shadow showed no enlargement in chest X-rays.

Discussion

At no time during the fasting period was there any serious change in blood pressure or pulse in our patients. Also, during the zero-calorie diet, neither after one week nor after two weeks was there any change in the current curves of the EKG worthy of note, if an insignificant and variable decrease of the height of the T-Wave in the left precordial chest wall drainage is ignored. This agrees with a series of studies in which zero-calorie diets of various durations led to no pathological changes of blood pressure, pulse, or EKG [5, 8].

Our studies demonstrated therefore, that we are dealing with changes in myocardian contractility or the pump performance of the heart, in a zero calorie diet.

The externally measured pressure rise time, which stands in close relationship to the pressure rise time measured in the blood [16], demonstrates a significant increase with an increase in duration of the denial of nourishment, yet at all points time it lies in the normal range [12]. Because of its inverse relation to the $dp/dt_{max}$ [18], it goes along with a decrease of this parameter. The strain time and the expulsion time are frequency-dependent on the systolic cycle time in their duration. A lengthening of the strain time derived from the frequency dependence, can above all be seen as an indication of a decrease of the left ventricular myocardial function, especially with diminished afterload. In our studies, as a matter of fact, at the end of the second week the strain time in the entire group, as well as in 7 of the 9 patients, lay above this normal range, whereby the average arterial blood pressure had simultaneously dropped as a measure of the afterload.

The suppression of the expulsion time shows likewise that a decrease of the contractility of the left ventricle takes place with increasing duration of denial of nourishment. Also, at the end of the second week of treatment, it was reduced in the entire group in comparison with the initial value, with statistical significance, whereby at this time in 6 out of 9 patients, the value was below the normal range for the given frequency. From this suppression with simultaneous lengthening of the strain time, a decrease of capacity of the left ventricle can likewise be concluded.

The expelled fraction should be used as a contractility parameter only with restrictions, since it depends not only on the myocardial contractility, but is also influenced by changes in the pre-load and in the after-load. [7, 13, 14]. It is greater with increasing contractility, but also with increasing the pre-load or decreasing the after-load. Because the value of the expelled fraction is in the normal range before the beginning of treatment, and is still normal at the end of the first week of the denial of nourishment, a clear decrease is calculated for the end of the second week. Since at this time, the average blood pressure, again taken as a measure of the after-load, was significantly lower than at previous times, a reduction of the pump performance of the left ventricle can be concluded from this decrease of the expelled fraction. A decrease of the pre-load with consequent decrease of the expelled fraction would be in fact theoretically possible, since Alexander and coworkers [2] had found a reduction in blood volume as well as a decrease in heart size in the chest X-rays with long-lasting denial of nourishment, yet in the chest X-rays with find no change in the heart shadow in chest x-rays, so that we might assume that essentially no decrease of the pre-load occurred.

The contractility index is a useful indirect parameter for establishing the contractility of the left ventricle [15], since it is closely correlated with $dp/dt_{max}$. Because $dp/dt_{max}$ is influenced by changes in the contractility as well as by changes in the pre-load, the after-load, and the frequency, the relationships of the contractility index are of a somewhat complex nature. In fact, there exists a weak positive correlation of the contractility index with the pulse frequency [11], yet changes in the contractility index resulting from changes in the pressure rise time count not only against the frequency-determined change of the contractility of the left ventricle, since the externally measured pressure rise time according to studies of Weissler and coworkers [23, 24] as well as by Knapp and coworkers [12] are frequency-independent. The significant decrease of the contractility index at the end of the second week of treatment, is on the one hand determined by a lengthening of the pressure rise time, even though it is found in the normal range, but on the other hand by a decrease in the diastolic blood pressure, which in this connection serves as a measure of the after-load. On this basis, we consider the reduction of the absolute value of the contractility index as a change of the myocardial contractility of the left ventricle, largely accompanying a reduction of the after-load, which nevertheless still remains within the physiological framework.

If the changes in the individual systolic cycle times and the parameters of the myocardial contractility calculated indirectly from them are considered together, then a denial of nourishment of two weeks duration leads to a lengthening of the strain time, a shortening of the expulsion time, and a decrease in the expelled fraction, which in consideration of pulse frequency and after-load indicate a reduction of the myocardial contractility of the left ventricle.

Sometimes positive correlations are found between the $K^+$ concentration in the serum and the expulsion time, as well as the expelled fraction. The question now arises, whether the decrease in the $K^+$ concentration and the change in the cycle parameter are two results of fasting, independent of one another, or whether the shortening in the expulsion time and the decrease in the expulsion rate are a result of the decrease in the serum $K^+$. The two correlations alone do not permit an answer to this question. Speaking against a dependence of the change in cycle time on the decrease of the $K^+$ concentration, is the fact that the decrease of the serum $K^+$ is not significant and that no correlation could be deomonstrated between the change in the $K^+$ concentration and the change in the cycle parameters. On the basis of our studies, this question cannot be answered with certainty, when all is said and done.

In the course of a zero-calorie diet, severe complications have seldom been observed up to now, yet Garnett and coworkers [9] reported on a 20-year-old girl who died of ventricular fibrillation after a 30-week fasting diet. We can also report a case [19], in which a patient with a sound circulatory system experienced a severe cardiac event after 18 weeks of fasting. Also, in this case, no changes in the EKG had been perceived up to the onset of the event. In the two cases discussed, it was a question above all, of extremely long fasting periods, which could lead to a loss of essential amino acids, and thus to a destruction of myofibrils. In our patients, however, there took place only a relatively short fasting period of two weeks, so that the decrease in heart performance can scarcely be attributed to a loss of protein, since this can probably become of importance only with distinctly longer fasting cures [5].

On the basis of the results of our studies, we are of the opinion that, by themselves, controls of blood pressure and pulse frequency as well as EKG recordings of fat persons with sound hearts, are not sufficient to establish the harmlessness of a zero-calorie diet for the cardiovascular system. We are of the opinion that a total fasting cure does not comprise a harmless operation, and that before the start, and above all during the execution of therapy, the circulatory condition of the patient should be verified repeatedly.

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